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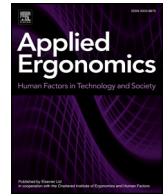
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Moving base driving simulators' potential for carsickness research

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ABSTRACT

We investigated whether motion sickness analogous to carsickness can be studied in a moving base simulator, despite the limited motion envelope. Importantly, to avoid simulator sickness, vision outside the simulator cabin was restricted. Participants ($N = 16$) were exposed blindfolded to 15-min lateral sinusoidal motion at 0.2 Hz and 0.35 Hz on separate days. These conditions were selected to realize optimal provocativeness of the stimulus given the simulator's maximum displacement and knowledge on frequency-acceleration interactions for motion sickness. Average motion sickness on an 11-point scale was 2.21 ± 1.97 for 0.2 Hz and 1.93 ± 1.94 for 0.35 Hz. The motion sickness increase over time was comparable to that found in studies using actual vehicles. We argue that motion base simulators can be used to incite motion sickness analogous to carsickness, provided considerable restrictions on vision. Future research on carsickness, potentially more prevalent in autonomous vehicles, could benefit from employing simulators.

1. Introduction

Motion sickness is a state of discomfort which can be caused by real or apparent motion (Reason and Brand, 1975). The underlying neural mechanism of motion sickness has been theorized to be a mismatch between actual and anticipated sensory signals, which can be modulated by visual-vestibular conflicts (Oman, 1990; Bles et al., 1998; Bos et al., 2008). Motion sickness can occur in multiple distinct forms including seasickness, carsickness, airsickness, and –more recently– forms involving artificial visuals such as simulator sickness and cybersickness (Golding, 2006b). Regardless of nomenclature, all such forms of motion sickness are understood as resulting from a similar mismatch in sensed and expected motion. However, there are also discernible differences between these forms. For example, seasickness, in addition to by definition occurring at sea, invariably involves a component of actual motion, i.e. external motion perturbation through ship movement (Lawther and Griffin, 1986). Conversely, in the case of cybersickness external motion perturbations are absent but the artificial visuals suggest motion leading to a visual-vestibular conflict, and subsequently to motion sickness (Davis et al., 2014).

Carsickness is motion sickness that results from provocative motion frequencies occurring in a road vehicle in transit, and can be exacerbated by mainly by visual factors (Turner and Griffin, 1999; Perrin

et al., 2013; Griffin and Newman, 2004a; Kuiper et al., 2018). The recent literature reports on comparatively few studies concerning carsickness (Kato and Kitazaki, 2006; Wada et al., 2012). Despite this limited interest, studies have indicated that about two-thirds of the population have suffered from carsickness at some point in their lives (Reason and Brand, 1975). Furthermore, autonomous vehicles, which are projected to become widespread in the coming decades, are expected to significantly increase the likelihood of carsickness (Diels and Bos, 2016). While the frequency dependency of provocative motion is reasonably well understood (O'Hanlon and McCauley, 1974; Lawther and Griffin, 1987; Bos and Bles, 1998) most data originates from experiments using vertical motion, which is subordinate to horizontal motion in cars (Griffin and Newman, 2004b). In addition, the functional role of visual-vestibular interactions in carsickness is not fully understood. Therefore, additional research into carsickness seems warranted.

Provided it is possible to reproduce specifically those motion cues that lead to carsickness, research into carsickness could benefit from utilizing moving base simulators. As opposed to on-road vehicle experiments, simulators offer a safe research environment and have the methodological advantage in their degree of controllability and replicability of motion and visual cues. Using simulators to investigate carsickness, however, firstly requires a thorough understanding of simulator sickness and secondly the prerequisite that the motion base can

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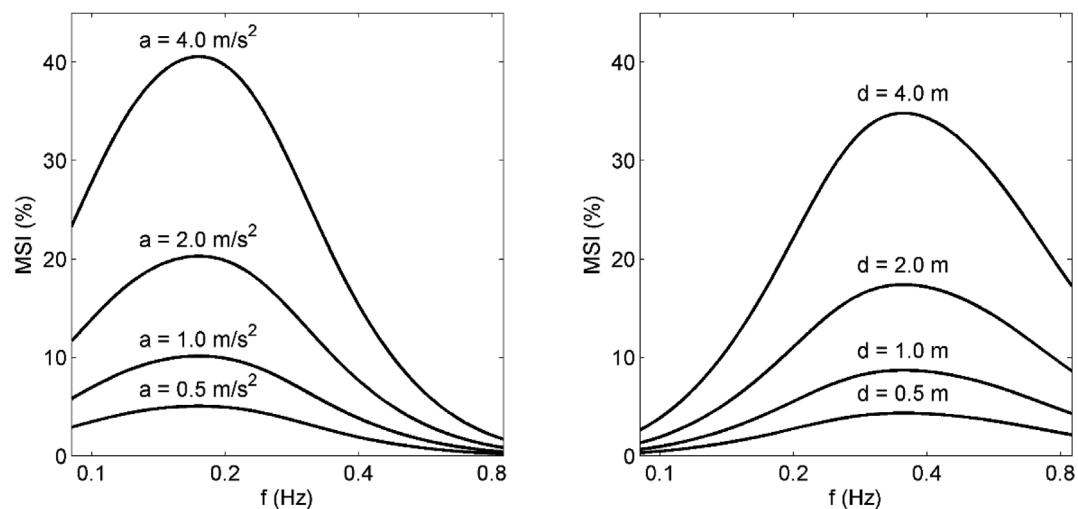


Fig. 1. Calculations based on ISO 2631-1(1997) showing the percentage of motion sickness incidence (MSI) depending on frequency sinusoidal motion lasting for 15 min. Left: calculations using fixed RMS accelerations (a). Right: calculations using fixed peak-to-peak displacements (d).

provide sufficient provocative motion to induce motion sickness. In the present study, we will discuss these two problems and investigate whether in a principal case a simulator can approximate car motion (i.e. the accelerations) of a sinusoidal motion resembling a slalom, and can induce motion sickness if visual factors are excluded.

Simulator sickness is commonly defined as motion sickness following any use of a simulator that leads to motion sickness (Hettinger et al., 1987; Brooks et al., 2010), and is primarily known as a practical problem causing participant drop-out when using simulators for training purposes (Reed et al., 2007; Mourant and Thattacherry, 2000). Typical simulator sickness can result either from *exclusively* the visual suggestion of motion, or from the *combination* of visual and vestibular cues. In a fixed base simulator, simulator sickness is somewhat akin to cybersickness (Hettinger et al., 1990), however, in a moving base simulator an interaction between visual and motion cues is at issue (Stanney et al., 1997). It should be noted that if a scenario leads to carsickness (e.g. a slalom), that scenario in a simulator also leading to sickness is not necessarily typical simulator sickness. Rather, it could be that the simulator resembles the real situation sufficiently that the motion sickness is caused by the same sensory conflict. However, due to the inherent difference between a driving simulator and a car in motion, it is very hard to identify the relevant sensory aspects that cause carsickness, and what (combinations of) sensory inputs lead to simulator sickness.

Therefore, to determine whether simulators can in fact be used to investigate carsickness, we would argue it should first be established without involvement of visual factors whether a moving base driving simulator can induce motion sickness. Visual cues are always central in simulator sickness, while for carsickness, mainly vestibular motion cues are at issue, possibly exacerbated by a visual-vestibular conflict (Kamiji et al., 2007). In fact, often precisely the lack of vision out-the-window aggravates carsickness (Griffin and Newman, 2004a; Kuiper et al., 2018). Thus, restricting vision out of the vehicle or simulator cabin during motion is compatible with carsickness, and even a naturally occurring facilitating factor.

In addition, if artificial visuals are present, differentiating what factors exactly cause sickness in a simulator is quite difficult (Kennedy and Fowlkes, 1992). The extent to which the artificial visuals lead to perceived self-motion and subsequent sickness depends on a plethora of factors, such as field-of-view, latency, depth or stereo vision, and contrast (Lin et al., 2002; Diels et al., 2007; Moss and Muth, 2011). While on the one hand, a larger field-of-view has repeatedly been shown to lead to increased sensation of self-motion (Allison et al., 1999; Van Emmerik et al., 2011; Grácio et al., 2014), visual information that is

excessively incongruent with expectations can potentially even be disregarded for self-motion perception, a phenomenon called ‘quarantining’ (Golding et al., 2009). For these reasons, restricting vision outside the simulator cabin prevents simulator sickness’ predominant visual component, and might allow study of sensory conflict as it would occur based on motion accelerations as they occur in a vehicle with no outside vision.

An additional issue with the use of driving simulators to investigate carsickness is their limited motion envelope, i.e., their limitations with respect to position, velocity and acceleration. Moving base simulators using a Stewart platform, for instance, are limited in their displacements, while xy-platforms offer a far greater range of motion. With respect to motion sickness, the frequency capabilities of the motion platform is of particular interest because motion in the frequency range around 0.2 Hz has been extensively shown to be most provocative for vertical (O’Hanlon and McCauley, 1974; ISO 2631-1, 1997). There is also evidence this is the case for horizontal motion (Golding et al., 2001). When using a motion base simulator to study motion sickness, to maximize provocativeness its frequency and acceleration capabilities should be carefully considered, as limited motion might not lead to any sickness to study (Golding, 2006b).

Somewhat counterintuitively, selecting a frequency of 0.2 Hz does not necessarily lead to the most provocative stimulus, if displacement is a limiting factor. Assuming a motion platform where the side-to-side displacement is the main limiting factor, that maximum displacement is a given parameter for a sinusoidal motion when maximizing provocativeness. Subsequently, the selected frequency is then directly related in magnitude to peak acceleration by the nature of a sinusoidal wave function. Following ISO 2631-1(1997), Fig. 1 shows that if freely selecting a frequency and maximum acceleration, the peak of sickness incidence is at about 0.2 Hz (left panel). Note that here displacement differs with frequency. However, if displacement is set, and thus frequency influences maximum acceleration, a frequency of 0.35 Hz maximizes expected sickness (right panel). This corresponds to a factor of 1.57 higher for 0.35 Hz compared to 0.2 Hz with the same amplitude. Thus, in order to maximize provocativeness for a set amplitude, a frequency of 0.35 Hz is expected to be ideal based on the ISO 2631-1.

In the present study we aimed to establish whether motion sickness analogous to carsickness can be induced using a simulator. To prevent simulator sickness, we exclusively use the simulator motion base, and excluded all visual cues by blindfolding participants. In addition, we aimed to establish what parameters for a sinusoidal motion would maximize motion sickness given the limited amplitude of the simulator. We compared two 15-min conditions. at 0.2 Hz and at 0.35 Hz and

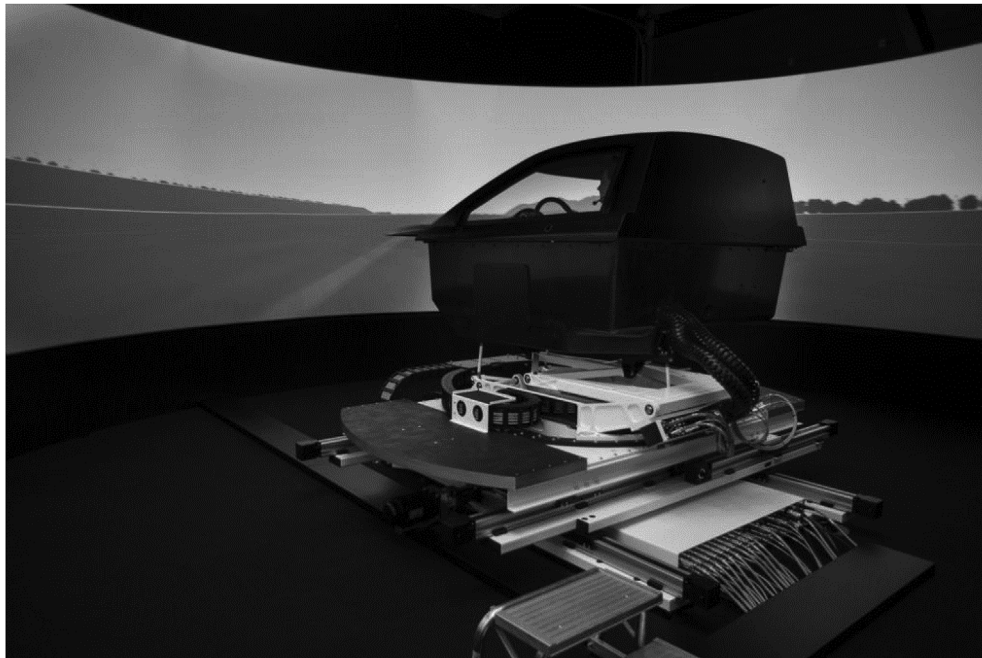


Fig. 2. The motion simulator. The partial vehicle cabin is lightweight and allows the x-y platform to smoothly move. For the present study we exclusively used lateral motion. Note that the visuals during the experiment were turned off, and the participant was blindfolded.

measured motion sickness with a self-reported scale every minute during the experiment.

2. Methods

2.1. Participants

Sixteen healthy adults voluntarily participated, 14 males and 2 females with a mean age of 37.31 years (SD = 13.5 years). All participants signed an informed consent form in advance, and indicated they were free of ocular and vestibular disorders and had normal or corrected-to-normal vision. All experimental procedures were conducted in accordance with the ethical guidelines of the Declaration of Helsinki.

2.2. Apparatus

The simulator was a moving base driving simulator consisting of a lateral sled on which a 6DoF motion platform was mounted with a car cabin. See Fig. 2. The maximum lateral displacement of the x-y platform was 100 cm from the center, i.e., 2 m peak-to-peak. However, the maximum displacement utilized in this experiment was 120 cm peak-to-peak to ensure a sinusoidal motion at the selected frequencies could be presented smoothly. The maximum lateral acceleration of the simulator was 7.4 m/s^2 . We did not make use of the simulator's additional motion capabilities in this experiment, thus all motion to which participants were exposed was lateral displacement on a single axis of motion. In terms of available lateral displacement, the simulator in the present study falls between two most common types of motion base simulators: those with only a Stewart platform (typically 20–100 cm peak-to-peak lateral displacement) and those with a linear track system in combination with a hexapod (up to many meters of lateral displacement).

2.3. Experimental conditions and stimulus

Two conditions were realized at two different frequencies in otherwise identical circumstances. These two conditions were 1) the 0.2 Hz condition, corresponding to a peak acceleration of 0.95 m/s^2 , and 2) the 0.35 Hz condition, corresponding to a peak acceleration of

2.90 m/s^2 . Each condition lasted for 15 min and lateral sinusoidal motion had an amplitude of 60 cm, i.e. 120 cm peak-to-peak. The stimulus was comparable in the relevant low frequency motion to slalom driving (Kuiper et al., 2018) or a continuous series of lane changes (Bellem et al., 2017).

2.4. Ratings

Prior to the first condition, participants filled out the motion sickness susceptibility questionnaire (MSSQ), adapted from Golding (2006a). The MSSQ gives an indication of a participant's susceptibility to motion sickness based on their past experiences. This was done to ensure that our population of participants was representative for the general population in terms of motion sickness susceptibility.

Our primary dependent variable was the MISC rating, an 11-point rating scale for motion sickness (MISC, also known as the misery scale, see Table 1, taken from Bos et al., 2005). This scale utilizes the fact that motion sickness is characterized by a multiple of symptoms such as sweating and dizziness, followed by nausea, retching and ultimately vomiting. Once the participant is familiar with this scale, employing it only takes a few seconds, i.e. the participant reports a single number when prompted. This allows for the scale to be easily applied repeatedly throughout the experiment.

Table 1
11-Point Misery Scale (MISC) (Bos et al., 2005).

Symptoms	Misc
No problems	0
Some discomfort, but no specific symptoms	1
Dizziness, cold/warm, headache, stomach/throat awareness,	Vague 2
sweating, blurred vision, yawning, burping,	Little 3
tiredness,	Rather 4
salivation,... but no nausea	Severe 5
Nausea	slight 6
	fairly 7
	severe 8
	(near) retching 9
Vomiting	10

2.5. Procedure

Conditions were counterbalanced across participants to compensate for order effects. After briefing, signing of informed consent, filling out the MSSQ, and explaining the MISC, participants took place in the front seat of the simulator cabin. Participants were seated upright, were blindfolded, and were presented with white noise over headphones. In this way only vestibular and proprioceptive cues differed between conditions. During the experiment, participants were prompted to report their level of motion sickness on the MISC scale (Bos et al., 2005) at 1-min intervals. Simulator motion was stopped when any level of nausea (i.e., MISC > 5) was reported, or after 15 min had passed, whichever came first. Having at least 24 h before the start of the next condition allowed participants to recover from any sickness in the previous condition, to further minimize any cross-over effects.

3. Results

The average MSSQ total score of participants was 11.20 ± 10.16 . This corresponds with a slightly below average susceptibility (Golding, 2006a). The 14 men had MSSQ scores of 12.67 ± 10.24 , while for the two females scores were relatively low (4 and 0.8). This is atypical as generally women are somewhat more susceptible (Dobie et al., 2001). MSSQ scores and motion sickness scores after 15 min were not significantly correlated for the two conditions ($r = 0.046$, $p = .877$ and $r = 0.003$, $p = .991$ for 0.2 Hz and 0.35 Hz respectively).

Motion sickness increased over the 15-min time period for both conditions. The average illness rating after 15 min was 2.21 ± 1.97 in the 0.2 Hz condition, and 1.93 ± 1.94 in the 0.35 Hz condition. Fig. 3 shows the illness ratings of participants for the two conditions over the entire 15-min period. A repeated measures ANOVA revealed a significant increase in illness score over time for both conditions ($F(1,195) = 11.872$, $p < .001$, partial $\eta^2 = 0.477$). However, there was no significant effect of the two conditions on illness scores ($F(1,195) = 0.249$, $p = .626$, partial $\eta^2 = 0.019$). In fact, there was a strong correlation between participants' MISC scores at $t = 15$ for the two conditions ($r = 0.770$, $p < .001$).

Regarding the percentages of participants over time that reached certain thresholds of illness rating (MISC) is another way to explore the

data. In most motion sickness studies, generally a portion of participants show no effect to the provocative stimulus (see e.g. Dong et al., 2011; Perrin et al., 2013). After 5 min, 75% of participants in our study reported initial motion sickness effects. During the entire 15-min exposure, 20% of participants did not report any illness symptoms in either condition (i.e. a score of 2 or more). Both of these trends can be seen in Fig. 4.

4. Discussion

In the present study we studied whether motion sickness, analogous to carsickness, here realized via a vestibular-proprioceptive conflict, can be induced using a moving base driving simulator. We blindfolded participants to ensure that no visual confounding factors were in play. We were successful in inducing motion sickness in three-quarter of participants. Both the fraction of participants reporting illness over the duration of the experiment and the overall severity of motion sickness were comparable to studies employing actual vehicles. These studies utilized, notably, similar provocative lateral accelerations, i.e. slalom of similar or larger amplitude (Kuiper et al., 2018; Wada and Yoshida, 2016). The percentage of participants in a 15-min timeframe reporting initial motion sickness symptoms in our study even exceeds that of a study using exposure to normal non-slalom drives for 30 min (Griffin and Newman, 2004a). Ratings we found were thus at least comparable in severity to on-road studies; our large fraction of male participants and lack of visual-vestibular conflict might have even led to potentially lower scores (Cheung and Hofer, 2002; Perrin et al., 2013; Kuiper et al., 2018). We therefore argue that a motion base driving simulator can in principle induce motion sickness analogous to carsickness, i.e. resulting primarily from low frequency motion.

While the velocity of a vehicle plays a large role in the driving experience, velocity has no direct bearing on our vestibular organs, pivotal in motion sickness. These organs are only sensitive to accelerations, i.e. changes in velocity (Mayne, 1974; Reason and Brand, 1975). In our experiment, the sensory input that leads to sickness was no different to the sensory input that principally leads to carsickness: low-frequency motion. This range of motion frequencies are, in a road vehicle, generally the result of acceleration and deceleration, cornering, and lane changes (Griffin and Newman, 2004b). Using the right motion

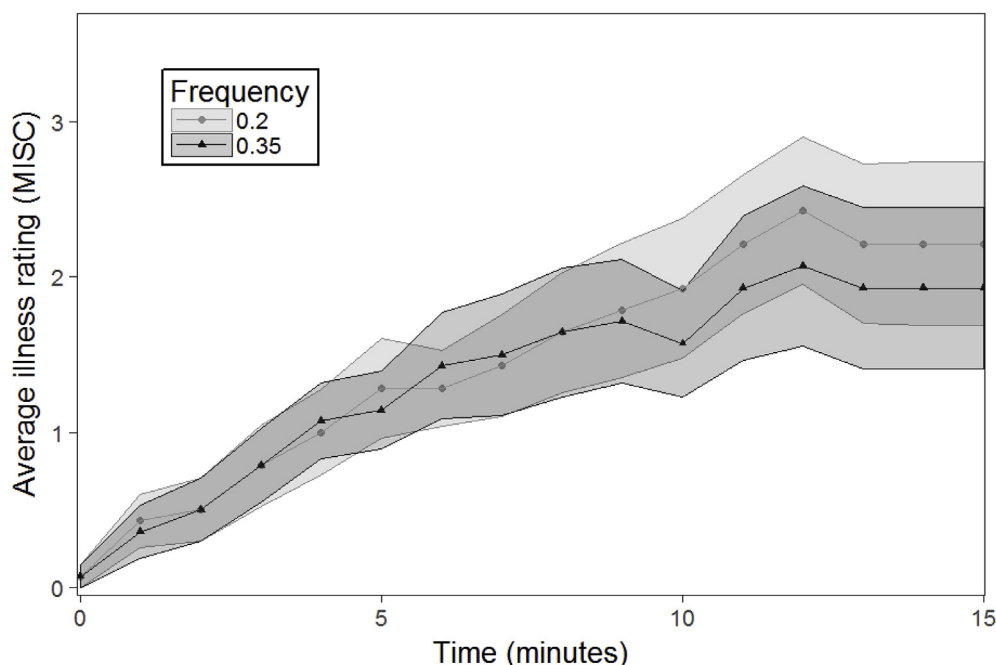


Fig. 3. Average illness ratings over time for the 0.2 Hz and the 0.35 Hz condition. Grey areas depict SEM.

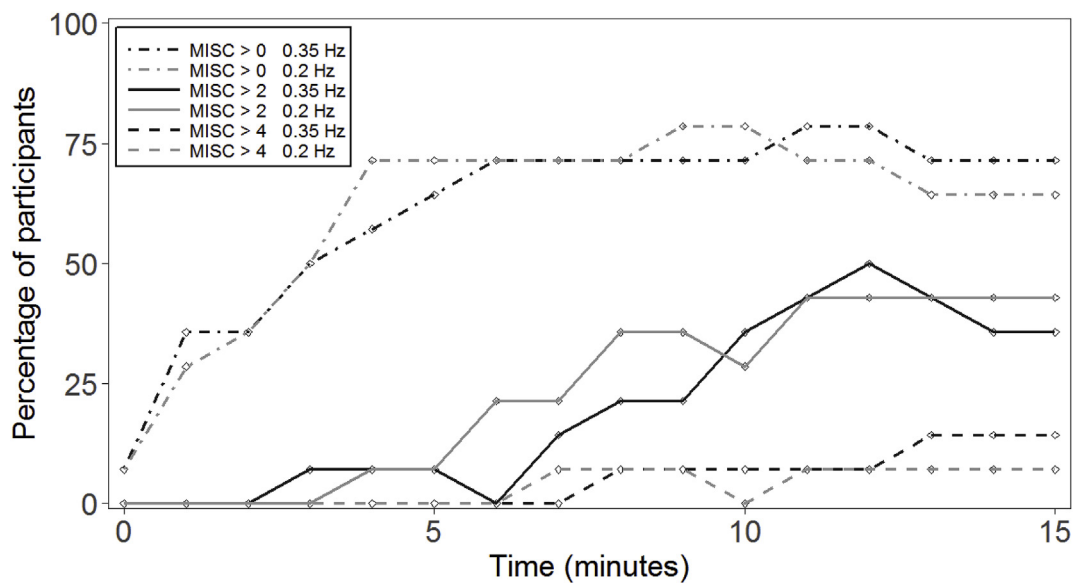


Fig. 4. Percentage of participants over time that reach a certain illness score or higher (MISC).

platform, the relevant frequency component of these motions cannot just be *simulated* but *recreated* in a simulator, thus potentially allowing researchers to apply exactly those motions which are principal to carsickness.

Assuming the same lateral displacement, we expected to find higher motion sickness scores at 0.35 Hz compared to 0.2 Hz, by a factor of 1.57 based the ISO2631-1. Although not statically significant, we observed a trend in the opposite direction. A possible explanation for these findings it that, while often generalized for horizontal motion, the ISO is based on vertical motion data. There is evidence that the frequency weighting for lateral motion, while also peaking at 0.2 Hz, is possibly distributed differently (Golding et al., 2001; Griffin and Mills, 2002; Donohew and Griffin, 2004). Motion frequency might play a larger role than peak acceleration in lateral motion as compared to vertical motion. An alternative explanation for the lack of difference between conditions is that in the 0.35 Hz condition the higher acceleration could have provided additional somatosensory cues via touch of the racing seat, or via vibration artefacts of the simulator, reducing illness. Overall, there is limited data available in the literature on the effect of motion frequency and acceleration for lateral motion, and our sample size was not sufficient to draw conclusions. More research on the relation between lateral motion and motion sickness is necessary, as lateral motion is the principal component of carsickness (Griffin and Newman, 2004a).

An advantage of researching motion sickness analogous to carsickness in a simulator is that it enables a wide variety of research that is potentially unsafe if preformed in a normal car, such as transfer of control in autonomous vehicles. Initially, such vehicles are expected to facilitate automated driving on select roads, with a moment of transfer of control back to the passenger when entering an area where automated driving is not supported (SAE, 2014). However, as passengers engage in non-driving activities during automated driving, their outside view is generally restricted, which exacerbates carsickness (Griffin and Newman, 2004a). Motion sickness has been found to degrade task performance (Rolnick and Bles, 1989; Bos, 2004), possibly degrading driving skills and thus creating unsafe situations if occupants are carsick. A second topic of research that could benefit from recreating carsickness in a simulator is that of countermeasures against motion sickness, i.e. providing addition sensory information to reduce sensory conflict and increase the ability to anticipate the motion (Rolnick and Lubow, 1991). Such measures have already been shown to be effective in both flight and ship simulators (Feenstra et al., 2011; Tal et al.,

2012), but have only very limitedly been investigated in cars (Miksch et al., 2016; Kuiper et al., 2018; Salter et al., 2019). In addition to visual information, there is evidence that auditory (Keshavarz and Hecht, 2014) or olfactory cues (Keshavarz et al., 2015), both easily implementable in a car interior or simulator cabin, can influence motion sickness.

Compared to blindfolded, vision on the cabin interior, such as when using a display for work or entertainment as one might do in an automated vehicle, could potentially increase the occurrence of sickness. This is due to increased visual-vestibular discrepancy as a result of the static visual scene (Probst et al., 1982; Bos et al., 2005; Griffin and Newman, 2004a,b; Kuiper et al., 2018). Research on the effect of reading or display-use during exposure to provocative accelerations is easily realizable in a simulator, and could test the effect of occupant behavior during automated driving at a moment when vision out of the simulator cabin is not relevant, thus avoiding the visual component of simulator sickness. Furthermore, these research paradigms can easily be expanded by including factors such as head position (Wada and Yoshida, 2016) or distraction (Bos, 2015). It should be noted that for carsickness, view on the car interior is more detrimental than having eyes closed, while vision out of the window is most beneficial (Probst et al., 1982; Griffin and Newman, 2004a,b; Wada and Yoshida, 2016).

Concluding, our findings suggest that moving base driving simulators have potential for studying motion sickness analogous to carsickness. We found motion sickness scores to increase over time at a similar rate as compared to on-road studies using similar motion stimuli. By restricting participants' vision, we excluded the visual conflicts associated with simulator sickness. It must be noted that researchers attempting to study carsickness should be vigilant that illness in a simulator is the result of a sensory conflict similar to carsickness, rather than of simulator sickness. In addition, the motion platform needs to be capable to generate accelerations equivalent to the relevant car accelerations. Within the constraints we mention, we believe simulators have potential to be used for carsickness research.

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